CASE REPORT

Beneficial effects of biventricular pacing in a patient with hypertrophic cardiomyopathy and intraventricular conduction delay

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The beneficial use of biventricular pacing is reported in a patient with hypertrophic cardiomyopathy and intraventricular conduction delay. This resulted in improvements in symptomatic status and exercise tolerance that may be related to cardiac resynchronisation. The improvement in symptoms by biventricular pacing in a patient with hypertrophic cardiomyopathy and intraventricular conduction delay is previously undocumented and requires further investigation.

We report the effects of biventricular pacing in a 63 year old man with hypertrophic cardiomyopathy (HCM) associated with significant symptoms of dyspnoea, chest pain, and palpitations. Previous cardiac catheterisation (November 1998) had shown normal coronary anatomy with a subaortic gradient of 77 mm Hg. Transthoracic echocardiography showed a non-dilated left ventricle (LV) with well preserved systolic contractile function, significant asymmetric septal hypertrophy (septal thickness of 2 cm), and a significant dynamic LV outflow tract (LVOT) gradient of 80 mm Hg. There was systolic anterior motion of the mitral valve and mild to moderate mitral regurgitation on colour flow Doppler ultrasound. There was evidence of diastolic dysfunction (abnormal relaxation pattern) with an E:A ratio of 0.9. The patient had undergone dual chamber pacing (atrial and right ventricular (RV) pacing) 18 months previously with limited symptomatic benefit. He remained significantly dyspnoeic with a limited exercise tolerance. His non-paced ECG showed significant intraventricular conduction delay (IVCD) with a QRS duration of 140 ms. Following insertion of a DDD pacemaker his QRS widened to 200 ms (fig 1).

We decided to implant an LV pacing lead in an attempt to reduce the IVCD and to improve his haemodynamic status. After full written informed consent was obtained, a dedicated unipolar left heart pacing lead (Uni Aescula LV model 1055K, Pacesetter Inc, Sylmar, California, USA) was implanted in January 2001 through the left subclavian artery and was manipulated through the coronary sinus to the middle cardiac vein. A DDDR pulse generator capable of simultaneously pacing both RV and LV (Chorum 7336B DDDR, ELA Medical, Newcastle upon Tyne, UK) was implanted in the left pectoral region. The patient underwent biventricular DDD pacing with a short atrioventricular delay of 78 ms.

Symptomatic status, exercise time, and echocardiographically derived haemodynamic parameters were assessed during biventricular pacing and compared with those during RV pacing with a short atrioventricular delay and no pacing. With biventricular pacing there was an improvement in symptomatic status, exercise time, and haemodynamics (assessed by echocardiography) over and above that produced by RV DDD pacing and far superior to no pacing (table 1, fig 2).

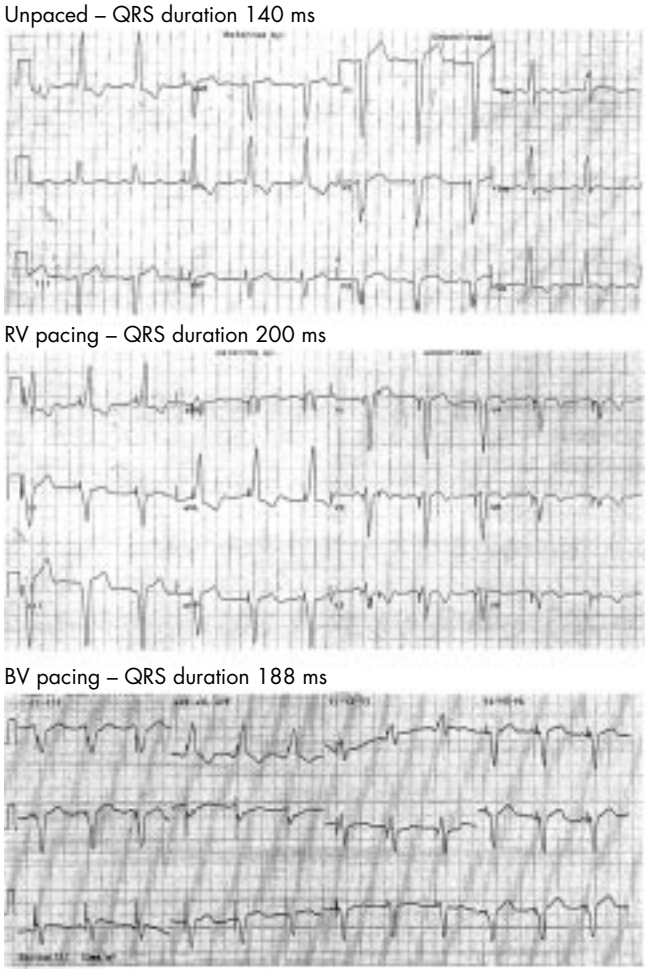

Figure 1 ECGs showing differing QRS durations before pacing, right ventricular (RV) pacing, and biventricular (BV) pacing.

DISCUSSION

The treatment of HCM has traditionally consisted of β blockers and calcium channel blockers. Surgical septal resection and more recently alcohol septal ablation have been used in HCM resistant to medical treatment. Permanent pacing in the form of dual chamber (DDD) pacing has been introduced as an alternative treatment option. By altering the pattern of ventricular depolarisation, pacing may result in a reduction in

Abbreviations: HCM, hypertrophic cardiomyopathy; IVCD, intraventricular conduction delay; LV, left ventricle; LVOT, left ventricular outflow tract; MUSTIC, multisite stimulation in cardiomyopathy; RV, right ventricle

Table 1 Changes in symptomatic status, exercise time, and left ventricular outflow tract (LVOT) gradient with different pacing modalities

	No pacing	RV pacing	Biventricular pacing
NYHA functional class	III	III	II
Symptomatic status	76	59	41
QRS duration (ms)	140	200	188
Exercise time (s)	228	180	358
LVOT gradient (mm Hg)	85	34	25

Symptomatic status was assessed using heart failure questionnaire. Exercise time according to standard Bruce protocol.

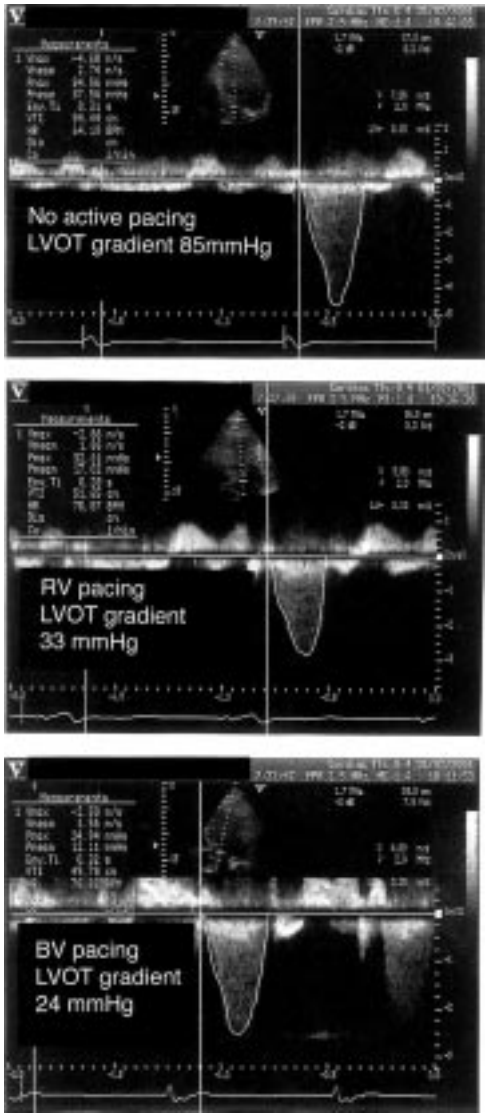

Figure 2 Reduction in dynamic left ventricular outflow tract (LVOT) gradient with pacing.

the LVOT gradient. Studies have shown reductions in LVOT gradients and functional status in patients with HCM resistant to medical treatment.[1] Benefits are similarly seen in patients without resting outflow tract obstruction, with improvements in exercise capacity and symptoms.[2]

The use of biventricular pacing in the treatment of severe heart failure is well documented. The basis of its potential benefit is that a significant number of patients with heart failure have IVCD characterised by discoordinate ventricular contraction and a wide QRS morphology.[3] Biventricular pacing can restore synchronous contraction and shorten QRS duration. Acute and short term improvements in haemodynamics have been shown in patients with poor LV function.[4 5] Uncontrolled studies suggest that biventricular pacing improves the condition of selected patients with heart failure (New York Heart Association functional class III to IV and QRS $>$ 150 ms).[6] The recently published MUSTIC (multisite stimulation in cardiomyopathy) study was the first major randomised study of biventricular pacing in heart failure and resulted in significant benefits in terms of exercise tolerance and quality of life.[7]

Our patient with HCM had significant IVCD, which was increased by RV pacing. Our rationale was to use biventricular pacing to achieve ventricular resynchronisation. Our results suggest an added benefit of an additional LV lead compared with RV pacing. Importantly, our patient was treated with a β blocker (metoprolol) and his drug regimen was not changed at any time either before or after pacing. There are, however, several limitations to our findings. Exercise tolerance was not measured objectively (oxygen consumption) before or after pacemaker implantation. Similarly, although we found changes in the resting LVOT gradient, we did not determine the status of the gradient provoked by manoeuvres such as Valsalva or vasodilators. There were no changes in systolic anterior motion of the mitral valve or mitral regurgitation with pacing but we assessed the severity of mitral regurgitation qualitatively and so small quantitative changes may have not been apparent.

Findings similar to ours have not been documented. To our knowledge biventricular pacing has not been used specifically in patients with HCM. This may be an important application and obviously requires further studies to document whether in general patients with HCM and broad QRS may benefit from cardiac resynchronisation.

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